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METHODS

Assessment of Pericardial Constraint: The Relation Between Right Ventricular Filling Pressure and Pericardial Pressure Measured After Pericardiocentesis

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Experimental studies have shown that right ventricular filling pressure (that is, intracavitary diastolic pressure) approximates pericardial surface pressure but, in many patients after removal of pericardial effusion, right ventricular filling pressure has been found to markedly exceed pericardial pressure recorded by an open catheter. The aim of this study was to determine whether this apparent contradiction was related to the technique of pericardial pressure measurement. Nine patients with chronic pericardial effusion were studied and, although these pressures diverged to varying degrees in individual patients, the previous observation was confirmed in that, although initially similar, right ventricular filling pressure and pericardial pressure (measured by means of an open catheter) tended to diverge during removal of the effusate; when the evacuation was as complete as possible pericardial pressure was 2.1 ± 1.0 (mean ± SE), while right ventricular filling pressure was 8.7 ± 1.7 mm Hg ($p < 0.01$).

In six open chest, anesthetized, volume-loaded dogs with pericardial effusion (50 ml), right ventricular filling pressure and pericardial pressures measured with both open catheter and flat balloon were all equal. With decreasing volume of pericardial fluid, right ventricular filling pressure and pericardial pressure (by catheter) diverged as had been observed in patients. However, pericardial pressure (balloon) continued to be equal to right ventricular filling pressure. (With 0 ml in the pericardium, right ventricular filling pressure = 12.9 ± 0.9 mm Hg, pericardial pressure [catheter] = 1.4 ± 1.9 mm Hg and pericardial pressure [balloon] = 12.4 ± 1.5 mm Hg.) Thus, these observations support the use of right ventricular filling pressure as an estimate of pericardial constraint in patients.

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In patients with ischemic heart disease it has been shown that the left ventricular diastolic pressure-volume relation may shift sharply after changes in ventricular loading conditions (1,2). Several animal studies (3–5) suggest that such changes in the left ventricular diastolic pressure-volume relation may be caused by changes in pericardial pressure. However, to date it has not been possible to evaluate this putative mechanism in patients since there is no accepted feasible means of estimating pericardial pressure.

In the normal and the failing dog heart, right ventricular filling pressure (that is, intracavitary right atrial and right ventricular diastolic pressures) was shown to be very similar to intrapericardial pressure recorded by a flat, liquid-containing balloon (Fig. 1) (6,7). Very recently, this relation between mean right atrial pressure and mean pericardial surface pressure was confirmed in patients intraoperatively (8). These studies suggest that right atrial pressure can be used to indirectly assess pericardial constraint. However, Kenner and Wood (9) found no relation between right atrial pressure and pericardial pressure (measured through an open catheter) when they caused the heart to enlarge by alternately obstructing the aorta and the pulmonary artery. Recently, Tyson et al. (10) supported the use of a protected micromanometer-tipped catheter to measure pericardial con-

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JACC Vol 7, No 2
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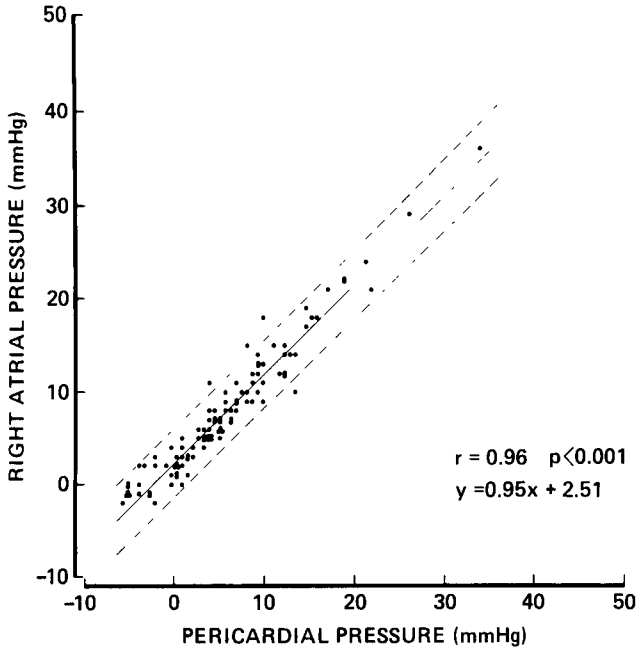


Figure 1. Right atrial versus pericardial pressure (recorded by balloon) in dogs. Recordings were obtained before and after induction of acute ischemic left ventricular failure. A wide range of pericardial pressures was obtained by volume loading and by intravenous administration of phenylephrine and nitroglycerin. Note that right atrial pressure approximated pericardial pressure. (Reproduced with permission from Smiseth et al. [7].)

straint. Furthermore, during withdrawal of pericardial effusion in patients, it was shown that, although right atrial and pericardial pressures (recorded by the drainage catheter) are initially similar, right atrial pressure is usually considerably higher than pericardial pressure after the pericardium has been emptied (10,11).

Because the discrepancies in these observations might reflect differences in methods of recording pressure, we attempted to determine whether the relation between right ventricular diastolic pressure and pericardial pressure during removal of pericardial effusion was dependent on the technique used to measure pericardial pressure. In patients with chronic pericardial effusion we compared right atrial pressure and pericardial pressure recorded through the drainage tube, while evacuating the pericardium. In dogs we compared right ventricular diastolic pressure with pericardial pressure recorded both by a flat, liquid-containing balloon and by an open catheter while the amount of pericardial fluid was varied.

Methods

Patient Study

Patients. The patient study group (Table 1) included nine patients (four men and five women, aged 34 to 71 years [mean 51]) who underwent pericardiocentesis for diagnostic or therapeutic purposes. In all patients written informed consent was obtained for the collection of the research data. The protocol had previously been reviewed and approved by the Joint University-Hospital Ethics Committee (January 1981). The cause of the pericardial effusion was malignancy in four patients, infective pericarditis in three and uremia in one. In the remaining patient the most likely cause was drug-induced pericarditis. An inspiratory decrease in systolic blood pressure greater than 10 mm Hg (pulsus paradoxus) was present in seven of the nine patients before removal of the fluid.

Pressure recordings during pericardiocentesis. Right heart catheterization was performed using a triple lumen, flow-directed catheter with the tip of the catheter positioned in the pulmonary artery. Right atrial pressure was recorded from the proximal port. Pericardiocentesis was performed from the subxiphoid approach. A 7 to 9F catheter with multiple side holes was advanced over a guide wire and utilized both to drain the fluid and to record pressure.

Patients were studied in the supine position with pressures referenced to the level of the midthorax. Mean right atrial and intrapericardial pressures were obtained using external transducers (model P23Ib, Statham-Gould, Inc.) and recorded with the standard lead II electrocardiogram (model VR16, Electronics for Medicine/Honeywell). After baseline values had been obtained, right atrial and intrapericardial pressures were recorded repeatedly after removal of 50 ml aliquots of effused fluid until no additional fluid could be withdrawn.

Animal Study

Animal preparation. Six mongrel dogs weighing 22 to 33 kg were used. Anesthesia was induced with sodium thiopental (Pentothal, Abbott Laboratories), 25 mg/kg body weight

Table 1. Patient Characteristics

Case	Age (yr)	Etiology of Pericardial Effusion	Removed Fluid (ml)	Pulsus Paradoxus	
				Before Removal of Fluid	After Removal of Fluid
1	54	Malignancy	350	+	–
2	70	Viral pericarditis	260	+	–
3	34	Malignancy	1100	+	–
4	47	Viral pericarditis	180	–	–
5	47	Viral pericarditis	350	+	–
6	38	Malignancy	810	+	–
7	52	Uremia	120	+	–
8	71	Drug-induced pericarditis	315	–	–
9	50	Malignancy	1250	+	–

intravenously, and maintained with halothane (1.5%) and nitrous oxide/oxygen using a constant volume respirator (Harvard Apparatus, model 607) and a closed rebreathing system. Left and right ventricular pressures were measured with 8F micromanometer-tipped catheters with reference lumina (model PC-480, Millar Instruments). A midline sternotomy was performed with the dog in the supine position. The ventral surface of the pericardium was incised transversely along the base of the heart. A flat balloon and an open-ended, multiple side hole catheter were positioned over the ventrolateral surface of the left ventricle at the mid-left ventricular level and loosely stitched to the epicardium. The open catheter was constructed by fitting a 4 cm terminal Silastic segment with an end hole and three side holes over the end of a 60 cm stiff 8F cardiac catheter. Another multiple side hole catheter was inserted into the pericardium to be used for drainage and saline infusion. The pericardium was sutured and sealed watertight by applying small amounts of glue (The Gripper Super Glue, Via Chem Inc.) along the sutures. A catheter was placed in a femoral vein for infusion of saline solution and withdrawal of blood, and another one was inserted into a femoral artery to monitor aortic pressure. Body temperature was maintained by a warming lamp. Pressures and the electrocardiogram were recorded at a paper speed of 75 mm/s (model VR16, Electronics for Medicine/Honeywell).

Pericardial balloon. The balloon was made from a folded sheet of Silastic (0.025 cm thickness) (Armet Industries Corporation) that was sealed at the edges; internally, the balloon measured 3 × 3 cm. A short Silastic tube (internal diameter 0.12 cm) protruding from the balloon cavity was connected to an 8F stiff cardiac catheter. When unconstrained, the balloon could hold up to 1.8 ml of fluid without developing measurable pressure.

Before the balloon was inserted into the pericardial space it was calibrated by a procedure similar to that described by McMahon et al. (13). The liquid-containing, bubble-free balloon was placed on a flat table and an air-filled plastic bag (about 10 liter capacity) was placed on top of the balloon. Pressure in the plastic bag was increased by direct manual compression (up to 20 mm Hg). Pressures measured from the flat balloon and the air-filled bag were displayed in an x-y fashion on an oscilloscope and the volume of liquid in the balloon (0.6 to 0.8 ml) was adjusted until the pressure recorded from the balloon equaled that in the bag over the entire pressure range.

The frequency response of the balloon was determined by placing the balloon in a test chamber. Pressure variations in the water (measured by a micromanometer-tipped catheter) were created by a pressure generator (models PC-480 and WGA-200, Millar Instruments). The pressure amplitude ratio (balloon/micromanometer) was 1.0 below 14 Hz and increased to 1.1 at 25 Hz. During the experiments the pressure signals were filtered above 25 Hz.

Experimental protocol. Blood volume was expanded by intravenous saline infusion until left ventricular end-diastolic pressure reached approximately 20 mm Hg; this level was maintained by a slow, continuous saline infusion. After a period of continuous pericardial suction, pressures were obtained (pericardium empty). Saline solution (to a maximum of 50 ml) was then infused into the pericardium at a rate of 7.6 ml/min using an infusion pump (model 607, Harvard Apparatus). Recordings were obtained with 50, 40, 30, 20, 10 and 0 ml of fluid in the pericardium.

Data analysis. Right ventricular mean diastolic pressure was compared with mean diastolic pericardial pressures, each averaged over a complete respiratory cycle. Right ventricular diastole was defined as the interval between the point of minimal diastolic pressure and the end of the *A* wave.

Statistics. In both patient and dog studies, data were compared statistically using Student's *t* test for unpaired samples (two-tailed). Differences were interpreted as being significant at a probability (p) value of less than 0.05. Values are given as mean ± 1 standard error.

Results

Clinical study. The amount of pericardial fluid removed ranged from 120 to 1,200 ml. Figures 2 and 3 show mean right atrial and mean pericardial pressures (measured by open catheter) during evacuation of the pericardium. In some patients (Cases 1, 2 and 3), the two pressures were initially similar but later, during withdrawal of fluid, right atrial pressure tended to exceed pericardial pressure. This was also true when the data from all patients were averaged in relation to volume of fluid withdrawn (Fig. 3). When the evacuation was as complete as possible, right atrial pressure was 8.7 ± 1.7 mm Hg and pericardial pressure was 2.1 ± 1.0 mm Hg. In other patients (for example, Case 9) both right atrial pressure and pericardial pressure decreased together. In each patient withdrawal of the pericardial fluid caused relief of signs of tamponade.

Experimental study. Pericardial pressures measured by open catheter and flat balloon were compared with right ventricular diastolic pressure while varying the amount of pericardial fluid. Figure 4 shows pressure tracings from a representative experiment; note that when the pericardium was empty, pericardial pressure recorded by the flat balloon approximated right ventricular diastolic pressure, whereas pericardial pressure recorded by the open catheter was markedly lower. Figures 4 and 5 show right ventricular pressure and pericardial pressures with varying amounts of pericardial fluid. Similar to the findings in patients (Fig. 2 and 3), right ventricular diastolic pressure exceeded pericardial catheter pressure when the pericardium was empty; right ventricular filling pressure was 12.9 ± 0.9 mm Hg, pericardial pressure recorded using the open catheter was 1.4 ± 1.9 mm Hg and pericardial pressure recorded using the

JACC Vol 7, No 2
February 1986 307–14

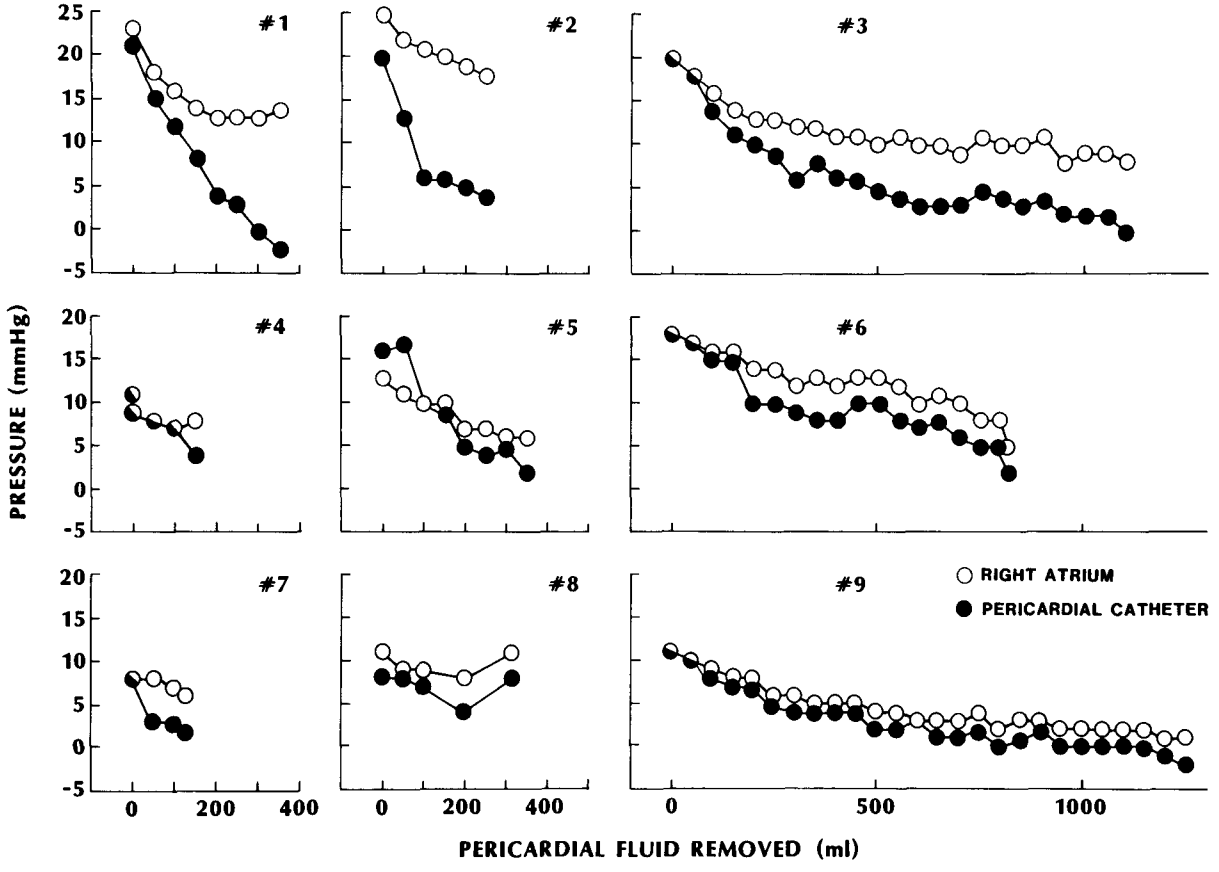


Figure 2. Individual data in nine patients showing right atrial pressure and pericardial pressure (recorded by means of an open catheter) during pericardial fluid evacuation. Note that with removal of pericardial fluid right atrial pressure exceeded pericardial pressure in most patients.

flat balloon was 12.4 ± 1.5 mm Hg. In two dogs the pericardial catheter recorded negative pressure when the pericardium was empty. With 30 to 50 ml of saline solution infused into the pericardial cavity, pericardial pressure recorded by both catheter and balloon approximated right ventricular filling pressure.

Discussion

Open catheter- versus balloon-derived pericardial pressure. The results of this study indicate that the recorded level of pericardial pressure and its relation to right ventricular diastolic pressure is dependent on the technique used to measure intrapericardial pressure. In the patients with pericardial effusion, right atrial pressure initially approximated pericardial pressure as recorded through the drainage catheter. During removal of the effusate, however, right atrial pressure and pericardial pressure diverged in some patients; when the evacuation was as complete as possible, mean right atrial pressure significantly exceeded mean pericardial pressure. (This divergence was not observed in all patients [see later].) These findings are in agreement with the clinical studies of Reddy et al. (11) and Grose et al. (12). In the experimental study we found a similar divergence between right ventricular filling pressure and pericardial pressure when the latter was recorded using an open catheter. With 30 to 50 ml (or more, presumably) of saline solution in the pericardium, pericardial (catheter) pressure was similar to right ventricular diastolic pressure and, as the pericardium was emptied, pericardial (catheter) pressure became much less than right ventricular diastolic pressure. However, the pericardial pressure recorded by a flat balloon approximated right ventricular diastolic pressure, regardless of the quantity of pericardial fluid. Thus, if pericardial (surface) pressure is properly measured there is no divergence from the value of right ventricular diastolic pressure. The apparent discrepancy arises only with respect to the measurement of pericardial liquid pressure, which our other studies (15) have indicated to be an inaccurate measure of pericardial constraint.

Pericardial "surface pressure" versus "liquid pressure." The difference in pressure measured by the open catheter and the flat balloon relates to the problem of measuring pressure between two opposing and compressing sur-

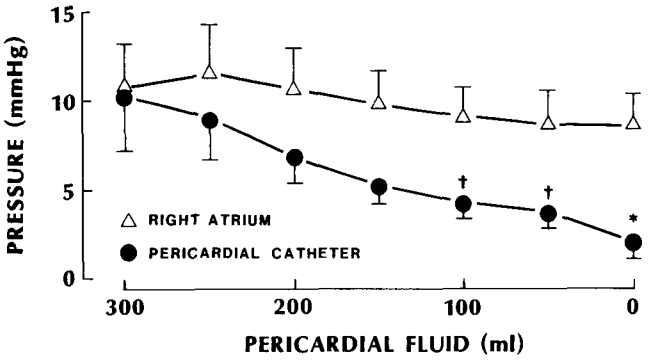


Figure 3. Patient data showing mean right atrial pressure and pericardial pressure (recorded by means of an open catheter) with various amounts of residual pericardial fluid. Maximal removal of pericardial fluid is represented by 0 ml. †$p < 0.05$; *$p < 0.01$.

faces. The balloon measures the normal force exerted by the pericardium per unit area of the balloon, that is, "surface pressure" (14,15). However, the open catheter measures only hydrostatic pressure, which may be less than surface pressure when the pericardium is unsealed or contains only a small (normal) volume of liquid (15). The difference between surface pressure and hydrostatic pressure represents the contact stress (16).

The fundamental difference between surface pressure and liquid pressure perhaps can be best appreciated by considering the analogous situation in the human knee. The liquid pressure as measured with a needle connected to a pressure transducer is normally near zero (17). However, the surface pressure, the average force per unit of common articular area, is immensely greater. Assuming that the weight of the head, the body and the thighs is 60 kg and the articular area of each knee is 3 cm^2 (18), the effective surface pressure is approximately 10 atm:

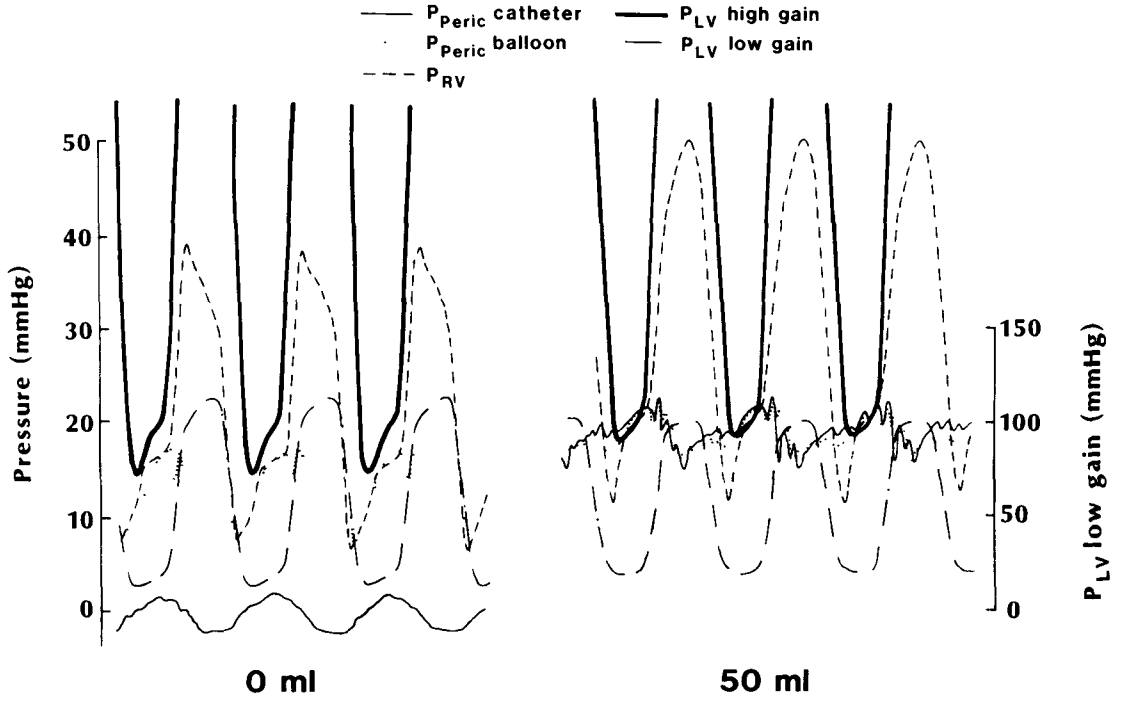
$$\left[\frac{60 \times 10^3 \text{ g} \times 980 \text{ cm/s}^2}{2 \times 3 \text{ cm}^2} \times \frac{1 \text{ mm Hg}}{1330 \text{ dynes/cm}^2} \times \frac{1 \text{ atm}}{760 \text{ mm Hg}}\right.$$

$$= 9.7 \text{ atm} \left.\right].$$

It seems obvious that the liquid pressure in the capsule is fundamentally unrelated to the mechanics of the joint. By analogy and on the basis of our recent study (15) (see later), we suggest that liquid pressure does not normally define pericardial constraint.

Acceptance that the flat, liquid-containing balloon provides a more accurate measure of pericardial constraint than does the open catheter is dependent on the validity of our theoretic approach. In our recent study (15) we postulated that the effective pericardial pressure (henceforth called "calculated pericardial pressure") must equal the difference between intracavitary left ventricular pressure (measured

Figure 4. Representative experiment showing the effect of pericardial fluid (50 ml) on pericardial and ventricular pressures. Pericardial pressure, recorded using a flat, liquid-containing balloon, approximated right ventricular diastolic pressure regardless of the presence or amount of fluid. However, pericardial pressure recorded using an open catheter was remarkably less than right ventricular diastolic pressure when the pericardium was empty. P_{LV} = left ventricular pressure; P_{RV} = right ventricular pressure; P_{Peric} = pericardial pressure recorded by open catheter and flat balloon, respectively.



JACC Vol 7, No 2
February 1986 307–14

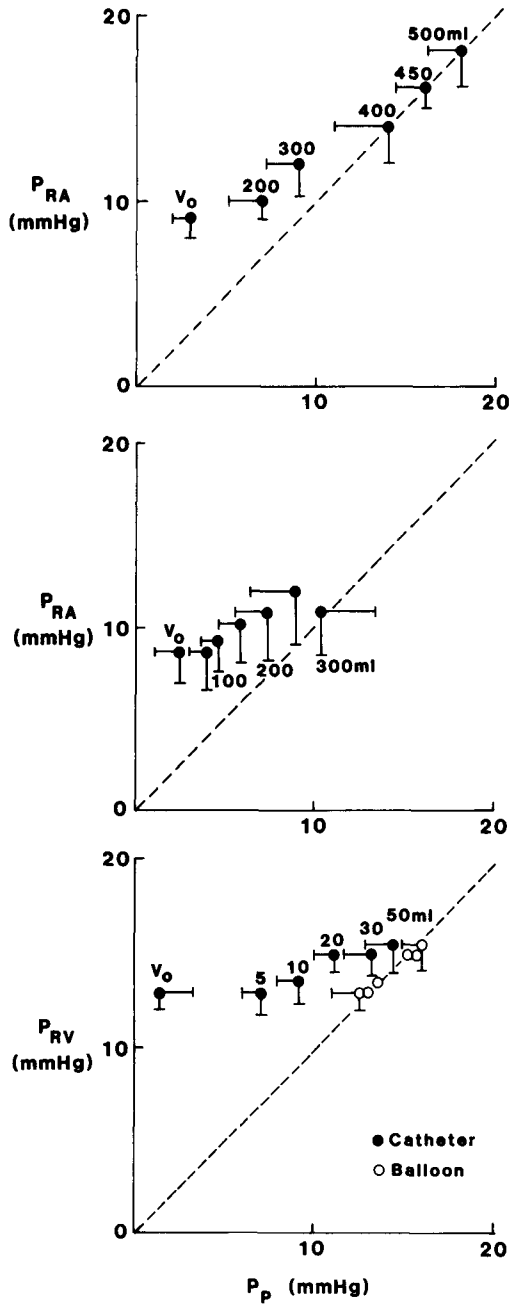


Figure 5. Relation between right ventricular filling pressure and pericardial pressure. The amount of fluid (ml) remaining in the pericardium (in excess of the minimal volume, V_0) is indicated. **Top**, Mean right atrial pressure (P_{RA}) versus mean pericardial pressure (P_P) (catheter). Data from Grose et al. (12) have been plotted with permission of the authors and the American Heart Association, Inc. **Middle**, Similar data from our clinical study. **Bottom**, Data from our experimental study show a similar relation as seen clinically between right ventricular filling (diastolic) pressure (P_{RV}) and pericardial pressure (P_P) (**solid circles**) measured with an open catheter (that is, liquid pressure). **Open circles** indicate that right ventricular filling pressure P_{RV} is an accurate indicator of pericardial constraint in that filling pressure was equal to pericardial surface pressure (measured with a balloon) regardless of the amount of fluid remaining in the pericardium.

with the pericardium intact) and the transmural left ventricular pressure measured at that volume. It was possible to measure transmural pressure directly at the end of the experiment by measuring intracavitary pressure with the chest open, the lungs retracted the pericardium removed after the circulating blood volume had been adjusted to allow the left ventricle to return to its original volume. This rationale is based on the assumption that a static equilibrium exists at end-diastole at the left ventricular endocardial surface such that intracavitary pressure is exactly opposed by the sum of transmural pressure and pericardial pressure. Using this rationale we demonstrated that pericardial pressure recorded by a flat, liquid-containing balloon accurately reflects pericardial constraint, regardless of the volume of pericardial fluid. The open catheter, however, recorded a pressure that was substantially lower than the calculated value when the pericardium was empty or contained less than 30 ml of fluid.

Tyson et al. (10) recently reported an experimental study in which pericardial hydrostatic pressure was recorded by a micromanometer-tipped catheter inside a fluid-filled pericardial catheter. In that it measures the hydrostatic pressure in the fluid, such a recording system is similar to the open-ended pericardial catheter used in the present study and differs only in that the frequency response of the micromanometer-tipped catheter is practically unlimited (2,500 Hz). In that study, however, the authors did not clearly describe how much fluid was present in the pericardium and therefore a comparison with the results in the present study is difficult. Furthermore, although the authors attempted to prevent scarring, given the unpredictable nature of the healing process after cardiac surgery and instrumentation, it is conceivable that sufficient liquid confluent with the micromanometer was trapped in a loculated space over the epicardium so that a force transduction similar to that produced by our balloon was effected. Finally, they did not systematically validate the recorded pericardial pressure by comparing it to another measure of pericardial constraint.

When the pericardium contains a substantial amount of effusate the heart is buoyed up by the surrounding fluid, so that the parietal pericardium and the heart are no longer in direct contact. There is then no contact stress and pericardial surface pressure equals the liquid pressure (that is, pericardial pressures recorded by the balloon and the open catheter are equal). However, with small amounts of pericardial fluid it has been shown (19) that the fluid layer is not uniformly distributed; the thickness of the fluid layer overlying the left ventricle is diminished at end-diastole. The mechanism for this may be a local rise in pericardial surface pressure at end-diastole due to stretching of the pericardium by the bulging ventricle. Because surface pressure then exceeds liquid pressure the fluid will move to a region where the pericardium is less stretched, probably along the atrioventricular sulcus. Thus, at end-diastole, pericardial liquid pres-

sure is probably less than surface pressure in some locations. When the pericardium is empty, liquid pressure will be minimal and may even become negative, depending on the amount of suction applied on the catheter. There still may be significant pericardial constraint, as is evident from our finding (15) that left ventricular end-diastolic pressure at a given ventricular volume was substantially greater when the pericardium was intact.

Similarity of right ventricular filling pressure and pericardial surface pressure. As shown in the present study as well as in previous studies (11,12) in patients, right atrial pressure sometimes remains abnormally elevated after the pericardial effusate has been removed. Our present animal data suggest that this is related to pericardial constraint. Manyari et al. (20) demonstrated a substantial increase in left and right ventricular size after evacuation of pericardial effusion in patients; this probably results from the increased circulating blood volume secondary to the prolonged reduction in cardiac output with tamponade. During evacuation of the pericardium the elevated venous pressure causes dilation of the right atrium and the right ventricle until intracavitary pressure is balanced by the sum of the transmural pressure and pericardial surface pressure. Because of the compliance of the right atrium and right ventricle, transmural pressures are low and practically unmeasurable; right atrial pressure therefore approximately equals pericardial surface pressure (Fig. 1) (7). The left side of the heart also dilates after pericardial drainage (20) but, because the left ventricle is less compliant, it develops a measurable transmural pressure and, therefore, left ventricular diastolic pressure exceeds pericardial surface pressure.

Because the diastolic pressures in the left and right ventricles are equal during tamponade, withdrawal of pericardial fluid (in our animal model) resulted in a larger decrease in diastolic pressure in the right than in the left ventricle. Since right ventricular diastolic pressure is essentially equal to pericardial pressure, this means that left ventricular transmural pressure initially increases with withdrawal of the effusion. We suggest that the same phenomenon occurring in humans offers an explanation for the observations of Reddy et al. (11) that hemodynamic improvement during pericardiocentesis stops when right atrial pressure and pericardial (liquid) pressure begin to diverge. With tamponade there is a high pericardial pressure and a low left ventricular transmural pressure (that is, a low effective preload). With pericardiocentesis there is a progressive decrease in pericardial pressure and increase in left ventricular transmural pressure. The left ventricle therefore enlarges in response to the increased effective preload and stroke volume increases. This increase in left ventricular effective preload continues only until pericardial surface pressure (right atrial or right ventricular diastolic pressure) declines to its minimum (plateau value), after which no further increase in left ventricular transmural pressure or stoke volume occurs (11).

Individual variation in the clinical study. While the experimental study showed a very consistent and marked divergence between pericardial (catheter) pressure and right ventricular filling pressure, there was some individual variation in the clinical data. However, our small series does seem to represent the spectrum of patients better described by Reddy and Grose and their respective collaborators (11,12). In many of their patients (and our Patients 1, 2 and 3), pericardial pressures recorded from the catheter and right atrial pressures diverged as the effusion was drained. On the basis of our experimental results and the striking observation noted by the former two groups of investigators, that hemodynamic improvement ceases when this point is reached, it seems clear that right ventricular filling pressure accurately reflected effective pericardial constraint in these patients (Fig. 5). In our experimental animals, right atrial pressure equaled pericardial surface pressure and in the previously studied patients ventricular performance improved only while right atrial pressure decreased; this strongly suggests that performance was directly related to end-diastolic volume according to the Frank-Starling relation, with end-diastolic volume being determined by transmural diastolic pressure which increased only as long as pericardial constraint continued to decrease. Another class of patients is represented by our Patient 9, whose right atrial pressure continued to decrease with pericardial liquid pressure as the effusion was drained. Since we do not have data on ventricular performance we can only assume that performance continued to improve as the left ventricle continued to dilate, as observed in the previous studies.

The considerable variation in these observations probably relates to how well the pericardium conforms to the shape of the ventricles. The first (divergent) pattern would be expected if, after the removal of a certain volume of effusion, the pericardium fit the ventricles rather well and this degree of constraint was unaffected by further removal of effusion. Such a situation might be found with an asymmetric effusion in which there is little fluid between the ventricles and pericardium. In contrast, the nondivergent pattern would ensue if the effusion were more uniformly distributed around the heart within a rather symmetrically distended pericardium. In this case, effective pericardial constraint would continue to diminish, causing the heart to expand until pericardial liquid pressure reached a minimum or until the heart expanded to fill the enlarged pericardium.

With respect to the lack of a simple relation between hemodynamic performance and the amount of fluid withdrawn, one must note that most of the decrease in volume probably can be accounted for by a decrease in pericardial volume, rather than by an increase in cardiac volume. The increase in cardiac volume is variable and essentially unpredictable quantitatively. Spodick and his collaborators (21,22) have also addressed the relation between the volume of pericardial effusion and the magnitude of the hemody-

namic alterations. They showed that the respiratory effects manifest during pericardial tamponade as pulsus paradoxus represent an exaggeration of the normal physiologic pattern and that similar hemodynamic alterations can be detected in patients with little or no anterior pericardial fluid. The possible implication of these studies—that pericardial constraint cannot be predicted from the volume of the effusion or even by the pericardial (liquid) pressure—is entirely consistent with our understanding that pericardial constraint is measured only by pericardial surface pressure.

Relevance of the present findings to effusive constrictive pericarditis. In a subgroup of patients with pericardial effusion there is significant constriction due to thickening of the parietal and visceral pericardium (23,24). This condition, which has been termed effusive-constrictive pericarditis, has been associated with the development of signs of constrictive pericarditis after evacuation of the effusion; right atrial pressure remains elevated and there is appearance of a prominent y descent while pericardial pressure (recorded by open catheter) approaches zero (24). Grose et al. (12) reported that right atrial pressure decreased to normal levels after surgical removal of a thickened parietal pericardium in such a patient. This suggests that before surgery with the pericardium evacuated pericardial constriction was present but was not (nor would be expected to be) reflected by the open-ended catheter measurement of pericardial pressure. In the present study, however, signs of tamponade disappeared after pericardial drainage in all patients without the development of hemodynamic evidence of constriction. Thus, it appears that thickening of the visceral pericardium was not an important factor in our patients.

Conclusion. The present investigation indicates that in the animal model right ventricular diastolic pressure approximates pericardial surface pressure (measured by a flat, liquid-containing balloon) regardless of the amount of pericardial fluid. We suggest that, when the pericardium contains no fluid or has been emptied of a substantial volume, the marked difference between right atrial pressure and pericardial pressure recorded by an open catheter reflects underestimation of pericardial constraint by the open catheter technique. These results support the use of right ventricular filling pressure as a useful approximation of pericardial surface pressure, thereby providing a means of estimating left ventricular transmural pressure changes in humans.

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